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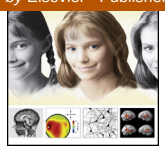

Commentary

Commentary on Spielberg at al., "Exciting fear in adolescence: Does pubertal development alter threat processing?"

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The adolescent brain has become somewhat of a "media darling," in part because of our voyeuristic opportunity to look under the hood to see its workings with recent advances in brain imaging, and in part because of the many seeming contradictions in teen behavior (Casey, 2013). At a time when teenagers may possibly be healthier than ever, we see a 200% increase in mortality (Dahl, 2001). Likewise, at a time when sensation seeking is at an all-time high (Steinberg et al., 2008), we see a peak in the internalizing disorders of anxiety and depression (Merikangas et al., 2010). Dahl and colleagues offer a provocative account for these conundrums in adolescent behavior for which they suggest there is supporting neuroimaging evidence. Their premise – teens find threatening situations exciting.

In support of their theory, the authors present hormonal and neuroimaging data from a longitudinal sample of adolescents who were scanned shortly before and after the onset of puberty. They report that pubertal increases in testosterone are associated with increased activity in both the amygdala and ventral striatum to threat cues (i.e., angry faces). The authors provide these imaging results as consistent with their theory given that amygdala activity is often seen in response to fearful stimuli, and ventral striatal activity is often seen in response to rewarding or "exciting" stimuli.

On the surface, thinking of adolescence as one big roller coaster ride of thrills in the face of potential danger seems like a tenable hypothesis. However, the imaging data provided to support their claims raise concerns in light of recent converging animal (Paton et al., 2006), human imaging (Delgado et al., 2008; Levita et al., 2009), and computational (Li et al., 2011) studies showing responses of these regions do not show strong valence specificity. For example, although amygdala activity has been shown in response to threat, it has also been observed in anticipation of rewards (Hommer et al., 2003), during presentation of neutral, happy (Somerville et al., 2004) and novel (Schwartz et al., 2003) faces and during self-induced sadness (Posse et al., 2003). Likewise, although ventral striatal activity has been associated with reward, it has also been observed to social (Britton et al., 2006), novel (Rebec et al., 1996) and even aversive stimuli (Levita et al., 2009).

Most recently, theoretical accounts have been put forth to account for the variety of stimuli to which the amygdala and ventral striatum respond. These accounts move away from simplistic one-to-one mappings of the ventral striatum and amygdala to positive and negative valence, respectively, toward the recognition of their distinct computational roles in learning (Li et al., 2011). Specifically, with the striatum playing a role in prediction error – the discrepancy between a predicted and actual outcome (Schultz et al., 1997; O'Doherty et al., 2003) and the amygdala playing a role in associative learning (Roesch et al., 2010), both of which influence adaptive action in response to positive and negative outcomes. Incorporation of this empirical and theoretical work may drive developmental cognitive neuroscience research more toward a mechanistic understanding of why adolescents engage in the behaviors they do.

In short, Dahl and colleagues present an intriguing theory of adolescent risk-taking based on the premise that adolescents perceive threats as thrilling. This theory is consistent with the neuroimaging data they present, but there are many alternative explanations, as well. Given the recent

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wave of cognitive neuroscience research indicating a lack of functional specificity between subcortical regions and specific emotions, neuroimaging analyses examining changes in activation in particular regions may have limited utility in testing emotion-based theories (Somerville et al., 2014). Stronger conclusions may be drawn through the use of formal reverse inference tools such as Neurosynth (Yarkoni et al., 2011) coupled with further testing of the theory using behavioral and psychophysiological approaches.

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